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Subadult Stress, Morbidity, and Longevity in Latte Period Populations on Guam, Mariana Islands

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ABSTRACT The frequency and age distribution of linear enamel hypoplasia (LEH) in the dentition of 293 individuals from Latte Period sites (AD 800–1521) on Guam, Mariana Islands, are examined in this study. Individuals dying as subadults (before age 16) and as young adults (ages 16–21) have more frequent LEHs than those who survived to middle or late adulthood, documenting a relationship between LEH-causing stress events and reduced life expectancy. The age distributions of cribra orbitalia and skeletal infection in children who died by age 10 exhibit striking similarities to the etiological age patterns of LEH in children, and those with skeletal infection have more frequent hypoplasias than children without infection. The comorbidity of systemic stress and infection in children, and their impact on life expectancy, are interpreted in the biocultural context of high population density in the large coastal villages of the late prehistoric period in the Marianas. Am J Phys Anthropol 104:363–380, 1997.

This study addresses the patterns of physiological stress observed in the skeletal and dental remains of subadults from Latte Period (AD 800–1521) villages on the west coast of Guam, largest of the Mariana Islands. The focus is on linear enamel hypoplasias (LEHs), their relationship to age at death, and their association with infection and anemia in children.

Enamel hypoplasias are defects in the dental enamel which appear as topographic irregularities on the tooth surface. They form as a result of temporary arrest in enamel matrix formation. The enamel forming cells, called ameloblasts, are sensitive to physiological stresses such as malnutrition, infectious disease, psychological or physical trauma, or other metabolic disruptions due to a wide variety of conditions (Kreshover, 1960; Pindborg, 1970; Sarnat and Schour, 1941). When the cycle of amelogenesis is disrupted, abnormally short enamel prisms are formed and are manifested as depressions, pits or grooves in the tooth surface. Hypoplasias remain visible until the affected enamel is worn away through dental attrition, providing a nearly permanent record of developmental arrest during infancy and early childhood. Although we cannot attribute LEH development to a specific disease or event in the life of a prehistoric child, studies of living children document the association between higher frequencies of LEH and poor nutrition and low socioeconomic status (Goodman et al., 1991, 1992).

As human life expectancy increases, there is a growing awareness of the long-term effects of suboptimal health in the fetus, infant and developing child: "the developmental lifespan perspective" (Henry and Ulijaszek, 1996). Undernutrition, infection, and

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stressors impair morphological development in children, and impact adult morbidity and mortality through metabolic imprinting and impaired immune response (Barker, 1994; Henry and Ulijaszek, 1996). And, as recently reviewed by Goodman (1996), a growing body of bioarchaeological literature links the prevalence of LEH to decreased life expectancy in prehistoric populations (Cook and Buikstra, 1979; Duray, 1996; Goodman et al., 1984; Goodman and Armelagos, 1988, 1989; Rose et al., 1978; Simpson et al., 1990; White, 1978). To paraphrase Goodman (1996), the challenge to the bioarchaeologist is to investigate both biological and cultural processes underlying the association between childhood stress and reduced life expectancy in prehistoric populations.

MATERIALS STUDIED

The data presented in this study are derived from the skeletal and dental remains of 293 individuals recovered in the course of cultural resource management projects by Paul H. Rosendahl, Ph.D., Inc. (PHRI) on Guam. Most of the individuals included in the study (220) are from the site of the Hyatt Hotel located on Tumon Beach on the west coast of the island. Between 1990 and 1993, 431 mortuary features were recorded at the 11-acre site. A minimum of 484 individuals, 42% of whom died before age 15 years, are represented in the entire Hyatt assemblage (Ryan, 1995). An additional 16 individuals are from the site of the Sandcastle Club adjacent to the Hyatt Hotel (Stodder et al., 1991a), and a small project at Gognga-Gun Beach near Tumon (Grant et al., 1992). Seven individuals are from the Mochom site, a large coastal community near Mangilao on the east coast of the island (Stodder, 1993). The remaining individuals are from two mitigation projects in the Agana Bay area: the Camp Watkins Project (n = 15) (Stodder et al., 1991b) and the Chaot/Agana Highway Project (n = 35).

The remains are poorly preserved; most individuals are incomplete and the bone is fragmentary. Natural and cultural disturbance factors at coastal sites in the Marianas include storm damage from typhoons and tidal surges, WW II activity, continued occupation and development in the beach communities, and prehistoric mortuary activities such as secondary burial, cremation, and partial disinterment for acquisition of long bones for tool manufacture and curation of skulls and other elements. The comparatively well-preserved dental remains were studied intensively as one means of maximizing data recovery from the fragmentary collections.

BIOCULTURAL CONTEXT

The Latte Period is named for the distinctive latte structures—parallel rows of monolithic upright stones topped by capstones, with each set of six to 12 latte stones supporting raised residences and other structures. In coastal settlements the latte sets are arranged parallel to the shoreline. Multiple extended interments are commonly found beneath the house or adjacent to the latte set perimeter, the individuals' feet pointing to the sea (Ryan, 1995; Thompson, 1932).

The Latte Period artifact assemblage is dominated by pottery, basalt mortars, shell adzes and other shell tools, bone awls, spear points, fishhooks and harpoons, slingstones, hammerstones, and of course latte stones (Grant et al., 1992; Hunter-Anderson and Butler, 1995).

Latte Period subsistence and settlement patterns are still being explored, particularly as the number of inland surveys increase our knowledge of noncoastal communities (Hunter-Anderson, 1997). Food remains document mixed horticultural and marine-based subsistence including yams, breadfruit, taro, coconut, seaweed, fish, shellfish, land crabs, and fruitbat. Isotopic analysis indicates a 20% to 50% marine protein component of diet, with very little contribution from terrestrial protein (Ambrose et al., 1997). Declining reliance on lagoon-based resources and increase in pelagic fishing and cultivated plants is indicated for the later prehistoric (Hunter-Anderson and Butler, 1995).

Environmental constraints on prehistoric productivity and health included limited availability of fresh water during droughts and invasion of water by storm surges (Hanson, 1990; Hunter-Anderson and Butler, 1995), and intermittent but not infrequent destruction of reef and garden areas, as well as tree crops, by typhoons. These may confi-

dently be assumed to have resulted in numerous short-term and seasonal incidents of undernutrition and increased endemic disease loads which might be expressed in the paleopeidemiological profiles of skeletal assemblages. As recently discussed by Brewis (1995), the traditional model of steady prehistoric population growth toward stasis as island resource limits are reached is contrary to the vulnerability and patterns of continual fluctuation of island populations, as indicated by the importance of interisland ties and resource distribution networks. Underwood characterizes the paleo- and historical demography of Guam as unstable and nonstationary within the context of long-term population growth. Population constraints were more likely in the form of sociocultural or political control than limited biotic resources (Underwood, 1973, 1997).

Cultural constraints are more difficult to define, since there is debate as to the nature of Latte Period social organization (Graves, 1986; Hunter-Anderson and Butler, 1995). Ethnohistoric accounts and archaeological reconstruction confirm at least a dual ranked society in the protohistoric, and suggest increasing definition of territoriality on the part of lineal descent groups. Height and size of latte structures have been equated with rank, and in the 1600s at least, the large coastal settlements seem to have been the more desired residential locations, inland communities the lower ranked (Driver, 1989; Hunter-Anderson and Butler, 1995). This suggests that the large communities on Tumon Bay may have been the residences of high ranking individuals or families. If so, then the skeletal remains studied here may represent the higher ranked elements of Latte Period Chamorro society on Guam. At issue, however, is whether all of the individuals interred beneath and around the latte sets actually lived in these houses and communities, or were simply buried there. Driver's (1983) treatment of the 1602 narrative of Fray Juan Pobre indicates that the dead were buried in front of the highest ranking relative's house—the head of the corporate descent group (Hunter-Anderson and Butler, 1995). Given the relative scarcity of burials at inland Latte Period sites, Hunter-Anderson (1997) has suggested that some inland residents were interred at coastal settlements.

Synthesis of the recent paleoepidemiological research in the Marianas is in the early stages, but some general characteristics of prehistoric health are mentioned here as background. Early European visitors described the Chamorros as robust and healthy (Driver, 1983). Stature estimates average 171.3 cm for males and 161.3 cm for females (Pietrusewsky et al., 1997) and sexual dimorphism is marked (Stodder, 1993). After nonspecific periostitis, treponematosis (endemic yaws) is the most frequent infection represented skeletally, reported at 8.8–25.6% in assemblages from the Tumon and Agana Bay areas (Pietrusewsky et al., 1997; Rothschild and Heathcote, 1993; Stodder et al., 1992; Trembly, 1996). Leprosy is documented in one burial from Saipan and in five individuals from the Hyatt site, dated as early as AD 830 ± 170 (Trembly, 1995). Gout is present in individuals from several assemblages on Guam (Rothschild and Heathcote, 1995; Stodder, 1993), and is thought to be indicative of high muscle mass (Houghton, 1996). Anemia indicated by cribra orbitalia or porotic hyperostosis on the crania is reported at low frequencies (Pietrusewsky et al., 1997) but subadult cranial remains are rarely preserved in Marianas assemblages, so the anemia rates are nearly all for adults. Caries are relatively rare in adult dentition, in part due to the cariostatic effect of chewing betel nut (Hanson, 1990, 1995). In contrast, rates of caries in deciduous dentition are very high (Hanson, 1988; Stodder, 1993). This may be attributable to local geomorphological factors affecting enamel quality and quantity such as high fluorine content in drinking water (Hanson, 1988; Sutton, 1978), or to consumption of sugarcane (Ambrose et al., 1997). In addition to fractures of the long bones, and bones of the hands and feet, spondylolysis in the lumbar vertebrae is commonly observed (Arriaza, 1997; Hanson, 1995; Pietrusewsky et al., 1997; Stodder et al., 1991b), the latter perhaps attributable to latte set construction activities.

Estimates of life expectancy in the Marianas assemblages vary widely, and many are of limited validity since the skeletal assemblages are often small. The theoretical

and methodological problems attendant to paleodemographic analysis are exacerbated in the Marianas by poor preservation, assemblages generated by the arbitrary excavation parameters of archaeological mitigation programs, and also by the problem of human remains recovered from disturbed nonfeature contexts. In many assemblages, the number of individuals recovered from mortuary features may be substantially lower than the minimum number of individuals (MNI) in the total assemblage from a site or locality (Stodder, 1993). Underwood's (1997) life expectancy/mean age at death estimates for the burials from Gognga-Gun Beach (22.5 years), Hyatt (22.9 years), and Apurguan (28.5 years) are most relevant to the present study, as they represent large coastal Latte Period settlements on Tumon and Agana Bays in the Tamuning District.

This study investigates the health of children in "downtown" prehistoric Guam. Tumon Beach was densely populated (Graves, 1991; Thompson, 1932). Burials from the Hyatt site were collected from 13 concentrations of mortuary features. Burial alignment and posthole patterns indicate a minimum of eight latte structures (Ryan, 1995). High fertility and substantial infant and child mortality rates are indicated for the Hyatt burials; the D30+/D5+ ratio is 55.95, and the D<1/D1–4 ratio is 68.18 (Underwood, 1997). High fertility and population density are important factors in community health and provide a framework for interpreting the subadult pathology data.

METHODS

All of the analysis was conducted in the PHRI Human Osteology Laboratory on Guam. Data presented here represent a very small portion of the PHRI bioarchaeological database for the Marianas. Only macroscopic, linear enamel defects—transverse grooves or rows of pits on the crown surface—are counted in these LEH data. Other enamel defects such as circular pits on the deciduous canines, circular caries, hyperplastic defects, and zones of discoloration are observed in the dental remains from the Marianas, but are not treated in the present study. Observation of LEH was limited by heavy calculus deposits on the premolars and molars, and by the Chamorro practice of incising cross-hatched or diagonal designs on the maxillary incisors of adults (Hanson, 1988; Leigh, 1929; Stodder, 1992, 1993).

The patterning of LEHs in the PHRI Guam assemblage is described in four sections of this paper: 1) LEH frequency in the deciduous incisors and canines; 2) the frequency of hypoplasia in all permanent teeth, recorded as part of standard PHRI dental data collection; 3) the mean number of defects per hypoplastic tooth, and 4) etiological age of hypoplasias in the permanent incisors and canines, estimated on the basis of their location in the tooth crown.

Presence or absence of LEHs was recorded for all the dentition as part of standard PHRI dental data collection. In addition, the location of each LEH observed in a permanent incisor or canine was recorded as distance from the cemento-enamel junction on the labial surface. The crown height of each permanent incisor and canine, including those with no LEH, was measured from the cemento-enamel junction to the incisal edge of the tooth. Measurements were taken with Helios dial calipers and recorded to the hundredth millimeter. The crown height indicates the developmental age range represented in the enamel remaining on each tooth, and thus the sample size of age-specific enamel segments in which the presence/absence of hypoplasias could be observed. Dental attrition is not extreme in the prehistoric Chamorro (Ikehara and Douglas, 1997; Leigh, 1929; Stodder, 1993), but the measurement corrects for occlusal wear and crown height reduction with age.

In order to estimate the age at which hypoplasias were formed—and thus the ages at which the physiological stress occurred—a population-specific chronology of enamel matrix formation was constructed. Following the method used by Swardstedt (1966), Goodman et al. (1980) and Stodder (1987), the location of each hypoplasia in the canines and incisors was converted to a developmental age using the standard enamel matrix formation chronology (Massler et al., 1941). Incisors and canines are used here because they have the greatest frequency of hypoplasias and the least amount of dental calculus,

TABLE 1. Crown heights (mm) of unworn incisors (I) and canines (C) from Guam skeletal assemblages

Tooth	Number	Minimum	Maximum	Mean
Maxillary I1	18	11.00	13.20	12.14
Maxillary I2	12	9.90	12.45	10.69
Maxillary C	12	10.80	13.00	11.81
Mandibular I1	14	9.20	10.95	10.11
Mandibular I2	14	9.40	11.20	10.35
Mandibular C	11	9.50	13.45	11.71

which obscures enamel in many teeth in the Guam assemblages. The developmental ages represented by enamel matrix formation in the permanent incisors and canines ranges from birth to 4 years in the mandibular incisors, birth to 4.5 years in maxillary incisors, and birth to 6 years in the canines. Each tooth crown is divided into enamel segments which correspond to 6-month age periods. The Guam Latte Period chronology for permanent incisor and canine development is shown in Figure 1.

The average heights of newly erupted and unerupted but complete crowns from Latte Period dental assemblages were used to adjust Swardstedt's chronology (based on a considerably smaller Medieval Swedish population) to the Guam dentition. As evident in Table 1, the sample of newly erupted or unworn teeth available for measurement is small. As larger numbers of dental remains from the Marianas are studied the chronology might be made more accurate, but as with other LEH chronologies, our accuracy is limited by the assumptions that the "timing of onset and completion of enamelization and the mean velocity of enamelization are known and exhibit no known variance" (Berti and Mahaney, 1992:20). As Goodman and Rose (1991) emphasize, these are best regarded as estimates of etiological age, to be considered in concert with other aspects of the paleoepidemiological profile.

RESULTS

Frequency of hypoplasias in deciduous anterior dentition

The deciduous incisors and canines of 134 children were included in the deciduous LEH frequency study. Seventeen individuals (12.69%) exhibit one or more LEH in an anterior deciduous tooth. Mandibular canines have the highest frequency of LEH: 9.43% (Table 2). LEH occurs in 5.18% of maxillary canines and in 2.98% of central incisors. All deciduous incisors pooled have a 1.26% frequency of LEH (six of 632). The canines pooled frequency is 4.49% (17 of 378). Children who died between ages 2 and 5 years have the highest frequency of deciduous LEH, except in the maxillary central incisors. LEH is absent in the incisors of any children over age 5 years, suggesting that deciduous LEH formation in the incisors is strongly linked to mortality in infants and young children in this assemblage. Of the 106 children dying by age 10 years, nearly 10% have LEH in the mandibular canine.

The overall (12.69%) and tooth-specific (0–9.43%) rates of deciduous LEH in the Guam sample are quite low compared to rates reported in children from prehistoric agricultural communities in North America: 23% in the Black Mesa Anasazi (Martin et al., 1990); 30% in the Protohistoric Zuni (Stodder, 1994); 45% in Woodland populations from the Lower Illinois Valley (Cook and Buikstra, 1979); 56% in Dickson Mounds Mississippians (Blakeley and Armelagos, 1985). Low socioeconomic status, poorly nourished children in highland Mexico (Goodman and Rose, 1991) and from the White Mountain Apache reservation in Arizona (Infante, 1974) have LEH rates of 14% and 19% in the incisors, respectively. The low rates of deciduous LEH typically seen in prehistoric Marianas assemblages (Hanson, 1988; Pietrusewsky et al., 1992, 1997) suggest relatively good pre- and perinatal physiological status.

Frequency of hypoplasias in permanent teeth

The relative frequencies of LEH in the permanent teeth are similar to those found in other studies (Goodman et al., 1980; Goodman and Armelagos, 1985; Stodder, 1987, 1994). The incisors and canines have the most defects. In the Guam assemblage hypoplasias are most frequent in the mandibular canines—24.26% (n = 280) exhibit one or more hypoplasia (Table 3). Of the maxillary central incisors, 22.26% exhibit hypoplasias, and 20.68% of the maxillary canines have hypoplasias. Mandibular molars have the fewest hypoplasias.

	Max I1		Max I2	Max C
Age at hypoplasia formation	0.0-----12.14	*Distance From CEJ (mm)*		0.0-----11.81
	0.5-----10.79			0.5----10.82
	1.0-----9.44		1.0-----10.69	1.0-----9.84
	1.5-----8.09		1.5-----9.16	1.5-----8.86
	2.0-----6.74		2.0-----7.64	2.0-----7.87
	2.5-----5.39		2.5-----6.11	2.5-----6.89
	3.0-----4.05		3.0-----4.58	3.0-----5.90
	3.5-----2.69		3.5-----3.50	3.5-----4.92
	4.0-----1.34		4.0-----1.53	4.0-----3.94
	4.5-----0.00		4.5-----0.00	4.5-----2.95
				5.0-----1.97
				5.5-----0.98
				6.0-----0.00

Mand I1	Mand I2	Mand C
0.0-----10.11	0.0-----10.35	0.0-----11.71
0.5-----8.85	0.5-----9.06	0.5-----10.74
1.0-----7.58	1.0-----7.76	1.0-----9.76
1.5-----6.32	1.5-----6.47	1.5-----8.78
2.0-----5.05	2.0-----5.18	2.0-----7.81
2.5-----3.79	2.5-----3.88	2.5-----6.83
3.0-----2.53	3.0-----2.59	3.0-----5.86
3.5-----1.26	3.5-----1.29	3.5-----4.88
4.0-----0.00	4.0-----0.00	4.0-----3.90
		4.5-----2.98
		5.0-----1.95
		5.5-----0.98
		6.0-----0.00

Fig. 1. Calibration chart for hypoplasias.

TABLE 2. Frequency of enamel hypoplasia in deciduous anterior teeth

	Infants (0–1.9 yr)		Children (2–4.9 yr)		Juveniles (5–9.9 yr)		Total (0–9.9 yr)	
Tooth[1]	Nw/N[2]	%w LEH[3]	Nw/N	%w LEH	Nw/N	%w LEH	Nw/N	%w LEH
Maxillary i1								
By individual	2/22	9.09	0/46	0.00	0/10	0.00	2/78	2.56
By tooth type	4/41	9.76	0/80	0.00	0/17	0.00	4/138	2.89
Maxillary i2								
Individuals	0/23	0.00	1/50	2.00	0/19	0.00	1/92	1.09
Teeth	0/40	0.00	1/84	1.19	0/32	0.00	1/156	0.64
Maxillary c								
Individuals	1/20	5.00	5/60	8.33	1/33	3.03	7/113	6.19
Teeth	2/37	5.41	7/98	7.14	1/58	1.72	10/193	5.18
Mandibular i1								
Individuals	0/17	0.00	1/57	1.75	0/11	0.00	1/85	1.18
Teeth	0/33	0.00	2/98	2.04	0/17	0.00	2/148	2.35
Mandibular i2								
Individuals	0/22	0.00	1/64	1.56	0/24	0.00	1/110	0.91
Teeth	0/42	0.00	1/110	0.91	0/38	0.00	1/190	0.53
Mandibular c								
Individuals	1/16	6.25	7/61	11.48	2/29	6.89	10/106	9.43
Teeth	2/24	8.33	13/10	11.93	3/52	5.77	18/185	9.73
Totals								
Incisors							6/632	1.26
Canines							17/378	4.49

[1] i = deciduous incisor; c = deciduous canine.
[2] Nw = number of individuals with one or more LEH; N = number of individuals observed.
[3] %w LEH = % of N with one or more LEH.

Table 3 also lists percentage of individuals with one or more LEH in either the right or the left antimere of each tooth. Thus the sample size for all maxillary central incisors is 301, but when the data are tabulated by individual the sample consists of 170 teeth. The *by individual* tabulation does not change the relative frequency of LEH per tooth type, but the samples are smaller, and resulting frequencies slightly different. Reports of enamel hypoplasia in Pacific populations typically present hypoplasia data tabulated by tooth type. However, in poorly preserved assemblages where many individuals are incomplete even in their dental remains, counting all teeth instead of one antimere per indi-

TABLE 3. Hypoplasia frequencies tabulated by tooth type and by individual

	By tooth			By individual		
	N[1]	Nw LEH	%w LEH	N	Nw LEH	%w LEH
Maxillary						
I1[2]	301	67	22.26	170	41	24.12
I2	294	36	12.24	169	22	13.02
C	295	61	20.68	169	38	22.48
P3	284	26	9.15	158	18	11.39
P4	271	12	4.43	156	9	5.77
M1	385	29	7.53	210	17	8.09
M2	293	31	10.58	165	20	12.12
M3	115	7	6.09	72	5	6.94
Mandibular						
I1	279	29	10.39	151	17	11.26
I2	283	37	13.07	171	23	13.45
C	280	68	24.26	161	42	26.09
P3	276	20	7.25	154	14	9.09
P4	259	17	6.56	148	11	7.43
M1	396	12	3.03	220	7	3.18
M2	307	15	4.89	169	9	5.32
M3	138	2	1.45	86	1	1.16

[1] N = number of individuals observed; NwLEH = number of individuals with one or more LEH; % w LEH = % of N with one or more LEH.
[2] I = incisor; C = canine; P = premolar; M = molar.

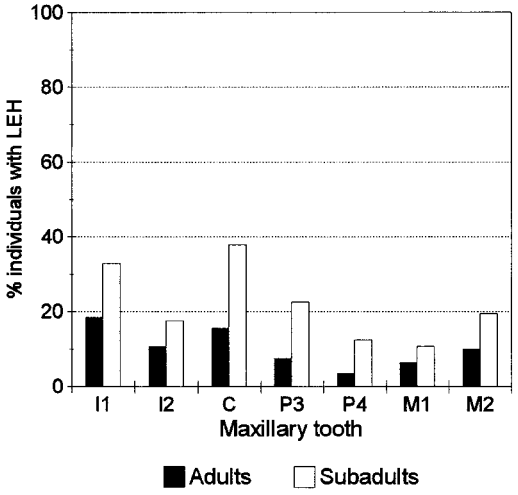

Fig. 2. Hypoplasia frequencies in adult vs. subadult maxillary teeth.

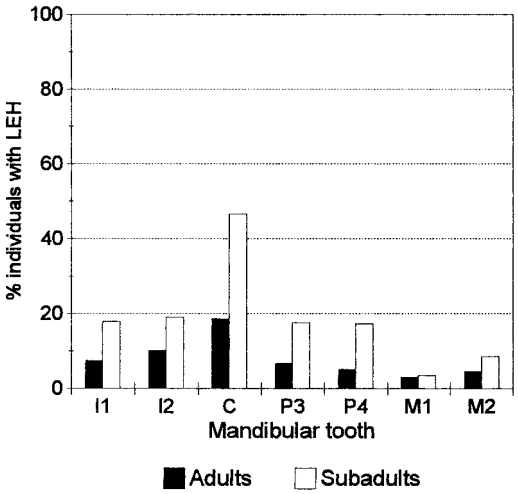

Fig. 3. Hypoplasia frequencies in adult vs. subadult mandibular teeth.

vidual may create a bias toward well-preserved individuals, as those with more teeth contribute more to the data set than those with only the right or left preserved.

In this assemblage, subadults (individuals who died before age 16) have consistently higher frequencies of hypoplastic teeth than individuals who lived into adulthood (Figs. 2 and 3). Percentage differences average about 11%. Subadult LEH frequencies are significantly higher ($P < .05$) in the upper and lower canines, the maxillary first incisor and the maxillary third premolar (Table 4). In populations with highly abrasive diets, such a pattern might be considered an artifact of attritional wear; the older the individual, the more dental enamel is worn away, and hypoplasias with it. However, dental attrition in Marianas skeletal populations is minimal until about age 40 (Leigh, 1929; Stodder, 1993).

A finer breakdown of the sample into children aged 2–4.9 years, juveniles 5–9.9 years, adolescents 10–15.9 years, young adults 16–20.9 years, and adults over 21 years of age highlights the striking decline in LEH frequency that occurs after age 20 (Fig. 4). The higher LEH rates in young vs. middle aged and old adults are statistically significant ($P < .05$) in the maxillary central incisor, the canines, maxillary third premolar, mandibular third and fourth premolars, and first and second molars.

Mean number of hypoplasias per tooth

The number of defects in each incisor and canine with hypoplasias was averaged for the different age groups in the sample. As shown in Table 5, subadults have a greater number of defects per tooth than adults, suggesting that the multiple occurrence of stress incidents is an important dimension of mortality in children. Adults have consistently lower mean numbers of defects than subadults. The mean number of defects per tooth declines with age; young adults have fewer LEHs than subadults; middle-aged and older adults have fewer than young adults. Mandibular incisors are the exception to the trend, but differences between the adult age groups are very small.

To summarize, in eight of 14 permanent teeth (third molars were not included in this portion of the study), the frequency of hypoplasias is significantly lower in people who lived until middle or late adulthood. Not only do the older age groups have a lower incidence of hypoplasia, they also have a smaller number of defects in the teeth affected than the individuals who died as subadults. These data strongly suggest that hypoplasias are related to age at death in the Guam Latte Period population.

TABLE 4. Hypoplasia frequencies in Hyatt subadults and adults

Tooth	Subadults Nw/N[1]	Subadults %w LEH[2]	Adults Nw/N	Adults %w LEH	Chi-square	Significance
Maxillary						
I1[3]	22/67	32.84	19/103	18.45	3.84	0.05
I2	10/57	17.54	12/112	10.71		
C	20/53	37.73	18/116	15.52	22.85	0.001
P3	9/40	22.50	9/118	7.63	5.16	0.05
P4	5/40	12.50	4/116	3.45		
M1	9/84	10.71	8/126	6.35		
M2	7/36	19.44	13/129	10.08		
Mandibular						
I1	10/56	17.86	7/95	7.37		
I2	12/63	19.05	11/108	10.08		
C	20/43	46.51	22/118	18.64	11.29	0.001
P3	6/34	17.65	8/120	6.66		
P4	5/29	17.54	6/119	5.04		
M1	3/87	3.45	4/133	3.01		
M2	3/35	8.57	6/134	4.48		

[1] Nw = number of individuals with one or more LEH; N = number of individuals observed.
[2] %w LEH = % of N with one or more LEH.
[3] I = incisor; C = canine; P = premolar; M = molar.

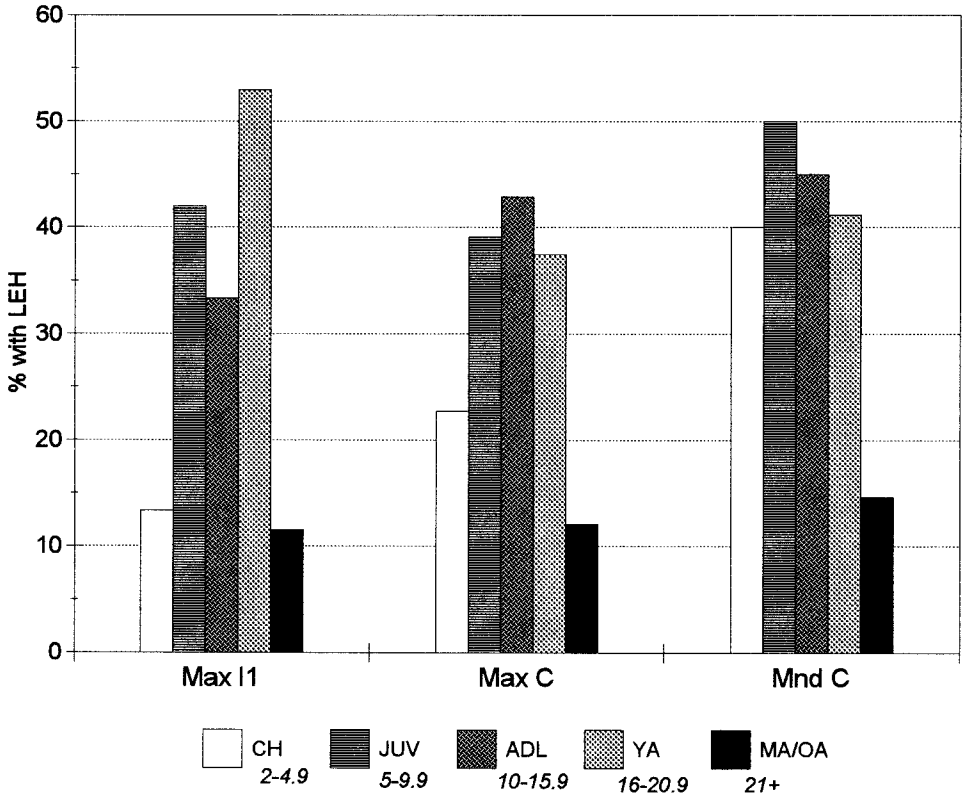

Fig. 4. Hypoplasia frequencies by age group.

The etiological age distribution of hypoplasias

One critical aspect of etiological age distribution of hypoplasias is the inherent, consistently observed, difference in age-specific susceptibility of the canines and incisors to the disruption of amelogenesis: incisors form hypoplasias earlier, with a peak at age 1 or 2

TABLE 5. Mean number of hypoplasias in incisors and canines

	Juveniles (5–9.9 yr)	Adolescents (10–15.9 yr)	All subadults (2–15.9 yr)	Young adults (16–20.9 yr)	Middle- aged and older (21+ yr)	All adults (16+ yr)
Maxillary I1						
Mean	1.07	0.90	0.86	0.77	0.24	0.33
N	14	10	28	13	51	67
S.D.	1.39	0.83	1.16	0.97	0.67	0.76
Maxillary I2						
Mean	0.82	0.56	0.64	0.23	0.15	0.16
N	11	9	22	13	52	69
S.D.	1.27	0.83	1.07	0.97	0.67	0.47
Maxillary C						
Mean	0.82	0.56	1.23	0.92	0.29	0.41
N	9	11	22	12	55	73
S.D.	1.23	1.14	1.13	1.04	0.65	0.77
Mandibular II						
Mean	0.23	0.42	0.30	0.08	0.11	0.10
N	13	12	27	13	46	62
S.D.	0.42	0.49	0.46	0.27	0.37	0.35
Mandibular I2						
Mean	0.37	0.93	0.62	0.18	0.20	0.19
N	13	11	27	11	54	68
S.D.	0.61	0.96	0.79	0.39	0.59	0.55
Mandibular C						
Mean	0.60	1.36	1.12	0.50	0.42	0.40
N	5	11	17	14	57	77
S.D.	0.80	1.23	1.13	0.63	0.72	0.69

I = incisor; C = canine.

years, and canines tend to form them later, with the typical peak at about age 4 years (Condon and Rose, 1992; Goodman and Armelagos, 1985). Presumably this is a function of the different morphology of these teeth, the different ages at which they develop, and other factors which are not well understood. The data are best presented and interpreted without combining tooth types.

Figure 5 shows the typical pattern of earlier peaks in hypoplasia formation in incisors and later peaks in the canines from the Guam sample. Three peaks are evident: at age 1 to 1.5 years, at 2.5 to 3 years and at 4 to 4.5 years. Few hypoplasias were formed in the earliest age segment—the first 6 months of life—and few at the end of enamel matrix formation period for the anterior teeth—age 5.5 to 6 years.

The elevated frequencies of hypoplasia in subadults is also demonstrated in the study of hypoplasias by 6-month enamel segments (Table 6). Hypoplasia frequencies are higher in subadults than adults in almost every age increment for every tooth studied. Part of the difference is an artifact of the smaller sample size for subadults, but the overall trend is supported by statistical significance ($P < .05$ or less) in the maxillary canines for ages 3.5–4 through 5.5–6, and mandibular canines for ages 5.5–6 and 6–6.5. Incisor differences are not significant. The adult and subadult patterns differ not only in the magnitude or overall frequency of LEH, but also in the ages at which LEHs were formed (Figs. 6 and 7). Individuals who died as subadults have more hypoplasias which were formed during early stages of incisor development and the later stages of canine development. In adults the frequency of defects in the canines increases to the normal peak at 4 to 4.5 years and then declines. In subadults, the peak is extended.

COMORBIDITY PATTERNS IN LATTE PERIOD CHILDREN

The frequencies of cribra orbitalia and skeletal infection in the subadults are listed in Table 7, and the age distributions in 0–10-year-olds are illustrated in Figure 8 with the LEH patterns from the teeth which match most closely—the maxillary central incisor and the maxillary canine. Each of these conditions has an age pattern which is partly determined by the nature of the stress indicator itself (such as the consistent tendency for canines to form hypoplasias later than incisors), by the limited age range

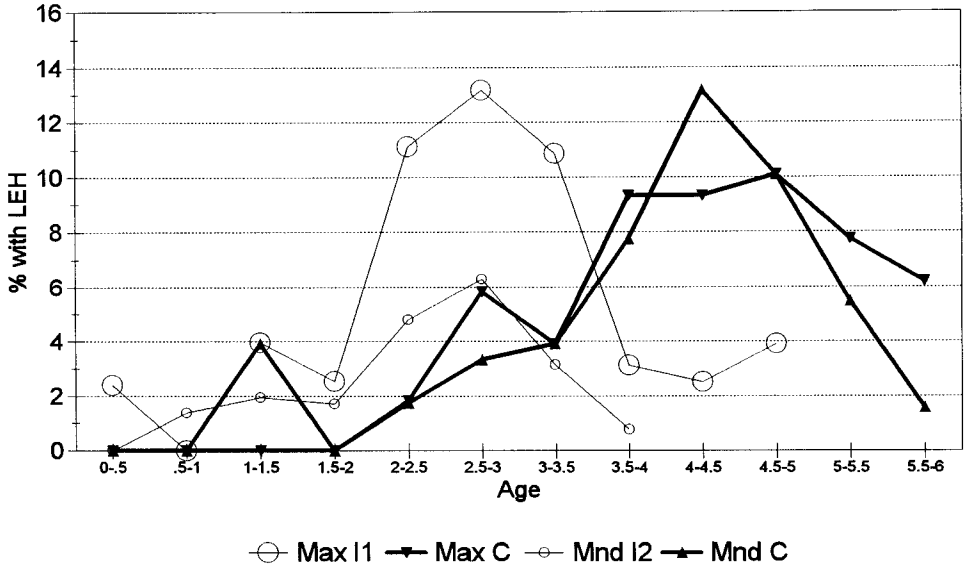

Fig. 5. Etiological age patterns in hypoplasia formation.

during which the stress indicator is recorded in the skeleton or tooth, and by the number and age distribution of individuals observable for each indicator. The composite paleoepidemiological profile in Figure 8 results from a series of estimates—estimated age at death, estimated age at hypoplasia formation, etc. Bearing these limitations in mind, there are clear correspondences in the patterns of LEH formation, anemia, and infection which suggest that the metabolic insults resulting in LEH formation influence and reflect multiple aspects of the developing child's health.

Cribra orbitalia

Unremodeled lesions of cribra orbitalia, which presumably represent active cases of anemia in subadults, are present in one or more orbits of 54% (18 of 33) of the individuals studied. There are so few intact orbits in subadult assemblages from the Marianas it is unknown whether this rate of anemia is typical for the region or not, but the presence of cribra orbitalia in all five of the 7-year-olds studied here seems unusual. Cribra orbitalia in infants and children aged 1 to 3 years is interpreted as reflecting anemia due to reduction of the infant's natural iron reserves, weaning stress syndromes, and early supplementation with high carbohydrate foods, a practice noted in the regional literature (Malcom, 1955). Between ages 2 and 5 years the rates of cribra orbitalia and LEH formation in the maxillary incisor follow quite similar patterns, suggesting common etiological factors. These might include parasitic infestation, a dietary iron deficiency, or inhibited utilization of iron due to infection or malnutrition. Selective eating habits and more far-ranging activity patterns are probable contributors to the anemia in older children, as is infection, skeletal evidence of which is present in 15% (two of 13) of the 7-year-olds.

Skeletal infection

Skeletal infection is present in 10% (24 of 248) of the subadults (Table 7). Half of the infections (13 of 24, or 54%) are nonspecific localized ($n = 8$) or systemic ($n = 5$) periostitis. Two infections (8%) are secondary to traumatic injury. Nine individuals (38%) exhibit proliferative nondestructive lesions, localized expansive nodes, or generalized diaphyseal expansion in the tibiae and other long bones typical of secondary yaws, as described by Hackett (1951). Minimum criteria for diagnosis of yaws included infection of at least one tibia, and occurrence of at least two types of diagnostic lesions on two skeletal elements (Stodder et al., 1992; Trembly, 1996).

TABLE 6. Frequencies of hypoplasias in 6-month enamel segments, adults and subadults

	Subadults			Adults			Total		
Tooth/age	Sw[1]	SN	S%w	Aw[2]	AN	A%w	Tw[3]	TN	T%w
Maxillary C									
0–0.5	0	13	0.00	0	17	0.00	0	30	0.00
0.5–1	0	18	0.00	0	30	0.00	0	48	0.00
1–1.5	0	21	0.00	0	51	0.00	0	72	0.00
1.5–2	0	22	0.00	0	74	0.00	0	96	0.00
2–2.5	1	23	4.35	1	87	1.15	2	110	1.82
2.5–3	1	23	4.35	6	97	6.19	7	120	5.83
3–3.5	1	23	4.35	4	105	3.81	5	128	3.91
3.5–4	6	23	26.09	6	106	5.66	12	129	9.30
4–4.5	4	23	17.39	8	106	7.55	12	129	9.30
4.5–5	6	23	26.09	7	106	6.60	13	129	10.08
5–5.5	6	23	26.09	4	106	3.77	10	129	7.75
5.5–6	6	23	26.09	2	106	1.89	8	129	6.20
Maxillary I1									
0–0.5	1	24	4.17	0	18	0.00	1	42	2.38
0.5–1	0	28	0.00	0	50	0.00	0	78	0.00
1–1.5	2	29	6.90	2	72	2.78	4	101	3.96
1.5–2	1	30	3.33	2	89	2.25	3	119	2.52
2–2.5	6	30	20.00	8	96	8.33	14	126	11.11
2.5–3	8	30	26.67	9	99	9.09	17	129	13.18
3–3.5	4	30	13.33	10	99	10.10	14	129	10.85
3.5–4	2	30	6.67	2	99	2.02	4	129	3.10
4–4.5	2	24	8.33	1	96	1.04	3	120	2.50
4.5–5	1	30	3.33	4	98	4.08	5	128	3.91
Maxillary I2									
1–1.5	0	17	0.00	0	23	0.00	0	40	0.00
1.5–2	0	24	0.00	0	63	0.00	0	87	0.00
2–2.5	1	24	4.17	0	80	0.00	1	104	0.96
2.5–3	3	24	12.50	3	96	3.13	6	120	5.00
3–3.5	1	24	4.17	3	97	3.09	4	121	3.31
3.5–4	5	24	20.83	7	97	7.22	12	121	9.92
Mandibular C									
0–0.5	0	9	0.00	0	18	0.00	0	27	0.00
0.5–1	0	12	0.00	0	42	0.00	0	54	0.00
1–1.5	2	14	14.29	1	63	1.59	3	77	3.90
1.5–2	0	15	0.00	0	83	0.00	0	98	0.00
2–2.5	1	16	6.25	1	99	1.01	2	115	1.74
2.5–3	1	16	6.25	3	104	2.88	4	120	3.33
3–3.5	0	16	0.00	5	112	4.46	5	128	3.91
3.5–4	3	16	18.75	7	113	6.19	10	129	7.75
4–4.5	3	16	18.75	14	113	12.39	17	129	13.18
4.5–5	4	16	25.00	9	113	7.96	13	129	10.08
5–5.5	4	16	25.00	3	112	2.68	7	128	5.47
5.5–6	2	16	12.50	0	112	0.00	2	128	1.56
Mandibular I1									
0–0.5	0	19	0.00	0	11	0.00	0	30	0.00
0.5–1	0	25	0.00	0	40	0.00	0	65	0.00
1–1.5	1	27	3.70	2	69	2.90	3	96	3.13
1.5–2	0	27	0.00	1	82	1.22	1	109	0.92
2–2.5	1	27	3.70	1	91	1.10	2	118	1.69
2.5–3	4	27	14.81	2	94	2.13	6	121	4.96
3–3.5	3	27	11.11	0	94	0.00	3	121	2.48
3.5–4	0	27	0.00	0	94	0.00	0	121	0.00
Mandibular I2									
0–0.5	0	20	0.00	0	17	0.00	0	37	0.00
0.5–1	0	25	0.00	1	47	2.13	1	72	1.39
1–1.5	0	27	0.00	2	76	2.63	2	103	1.94
1.5–2	0	27	0.00	2	90	2.22	2	117	1.71
2–2.5	2	27	7.41	4	98	4.08	6	125	4.80
2.5–3	4	27	14.81	4	100	4.00	8	127	6.30
3–3.5	3	27	11.11	1	101	0.99	4	128	3.13
3.5–4	0	27	0.00	1	101	0.99	1	128	0.78

I = incisor; C = canine.
[1]Sw = sub adults with one or more LEH in an enamel segment; SN = number of subadult enamel segments observed; 5%w = % of SN with one or more LEH.
[2]Aw = adults with one or more LEH in an enamel segment; AN = number of adult enamel segments observed; A%w = % of AN with one or more LEH.
[3]Tw = total individuals (adults plus subadults) with one or more LEH in an enamel segment; TN = total number of enamel segments observed; T%w = % of TN with one or more LEH.

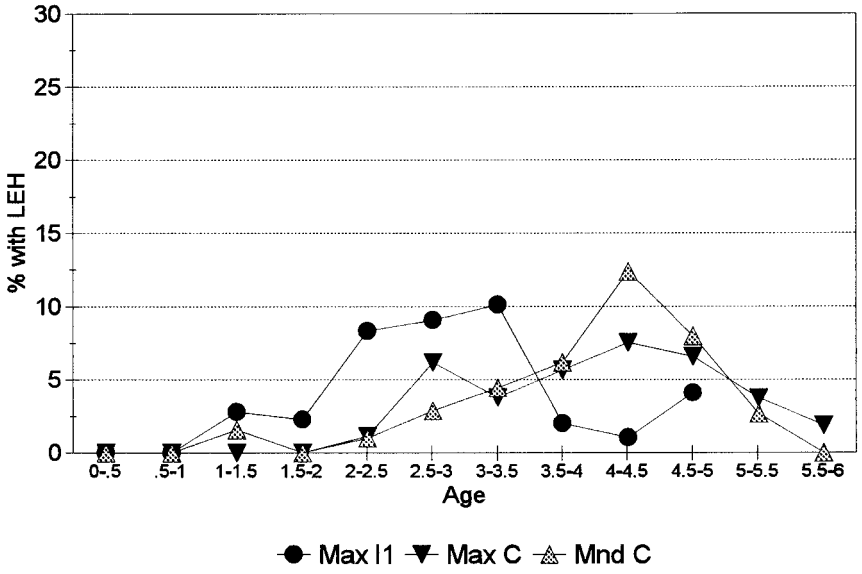

Fig. 6. Age patterns of enamel hypoplasias in adults.

Treponematosis frequencies of 19 to 25% in the Hyatt and Gogna-Gun Beach skeletal assemblages (Rothschild and Heathcote, 1993; Stodder et al., 1992; Trembly, 1996) suggest that yaws may have been hyperendemic in Latte Period villages on Tumon Bay. In communities with endemic yaws, the infection is contracted by virtually every child, starting as early as the first year of life in some Pacific island populations (Eason et al., 1985; Marples and Bacon, 1953). Transmission increases in toddlers and older children; new cases decline after adolescence. The infection is communicated predomi-

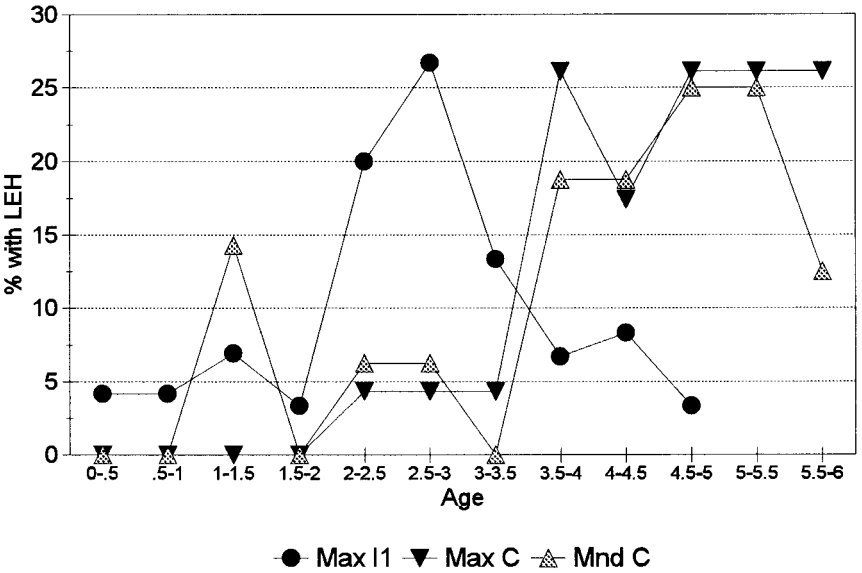

Fig. 7. Age patterns of enamel hypoplasias in subadults.

TABLE 7. Cribra orbitalia, infection and LEH frequencies in subadults

	Cribra orbitalia		Skeletal infection		Max I1 LEH		Max C LEH	
Age	Nw/N[1]	%w	Nw/N	%w	Nw/N	%w	Nw/N	%w
0	1/1	100	3/50	6	1/24	4	0/13	0
1	1/1	100	5/21	24	0/28	0	0/18	0
2	1/3	33	2/26	8	3/59	5	0/43	0
3	2/3	66	2/21	10	14/60	23	2/46	4
4	1/2	50	3/29	10	6/60	10	7/46	15
5	2/5	40	1/23	4	3/54	5	10/46	22
6	1/3	33	1/10	10			12/46	26
7	5/5	100	2/13	15				
8	1/1	100	0/7	0				
9	1/2	50	0/8	0				
10	0/2	0	1/12	8				
11	0/1	0	2/2	100				
12	1/2	50	1/8	12				
13			1/2	50				
14			0/3	0				
15	1/2	50	0/12	0				
Total	18/33	54	24/248[2]	10	27/285	9	31/258	12

[1] Nw = number of individuals with cribra orbitalia; N = number observed; %w = % of N with Cibra orbitalia.
[2] Includes one subadult of unknown age.

nately by skin contact among infected children (Hackett, 1957), the leg being the most common site of initial infection (Bruce-Chwatt, 1978; Hill, 1953). The primary stage, which does not affect the skeleton, is of quite variable duration and intensity. Primary yaws may resolve or advance to the secondary stage which can last as long as 5 years with intermittent latent periods. Tertiary yaws, which involves disseminated skeletal and soft tissue lesions (Bittner, 1926; Hackett, 1951), is present in 6% (all adults) of the current study population, a frequency consistent with the rate of tertiary yaws reported in epidemiological studies of yaws-endemic islands in the New Hebrides (Mills, 1955).

Treponematosis is present in infants, 5- and 6-year-olds, and 10- and 11-year-olds in this assemblage. The roughly concurrent increases of infection and LEH in children ages 5 to 6, and of infection and cribra orbitalia in 6- and 7-year-olds, coincide with the epidemiologically documented increase in treponematosis in children of this age group (Hackett, 1957). Infection in the adolescents probably represent cases of chronic treponemal infection, which may have had a latent phase after initial infection in infancy.

The proposed relationship between infection and LEH formation is supported when the frequencies of LEH are compared in children with and without skeletal infection. Children with skeletal infection have higher mean numbers of hypoplasias in their incisors and canines, and in all permanent teeth combined, than children with no skeletal infection (Table 8). The frequencies are significantly higher in the maxillary canine, the mandibular incisors, and in the pooled data for all teeth.

DISCUSSION

This study demonstrates that members of Guam Latte Period populations dying as subadults and young adults (before age 21) experienced more frequent events of hypoplasia-causing stress than those surviving into middle and old adulthood. Compared to those who lived into adulthood, those who died in childhood and adolescence experienced greater stress between birth and age 1.5 and between ages 4.5 to 6. Subadults have a higher mean number of hypoplasias per tooth than adults. The mean number of defects shows a fairly regular decline from youngest to oldest age groups. Children with skeletal infection have more LEHs. The patterns of comorbidity do not tell us whether the infections caused the hypoplasias and anemia, or alternatively, whether the stress incidents documented in the hypoplasias increased individual frailty and heightened susceptibility to future infection and early mortality.

Probably both processes—the synergism of physiological stressors and impaired immune function—were operating. This was in the context of a densely populated community whose subsistence base was subject to disruption by tropical storms as well as droughts, and in which some members may have been socially favored. Grave goods are rare in Latte Period burials, so we do not have an index of social status based on artifactual accompaniment. Some intrasite social differentiation is implied in the distinctive age and sex composition of certain burial clusters at the Hyatt site (Ryan, 1995). Comparative study of health between burial clusters is limited by small sample sizes, but future research will address the hypotheses that individuals interred in clusters with proportionately more children and women

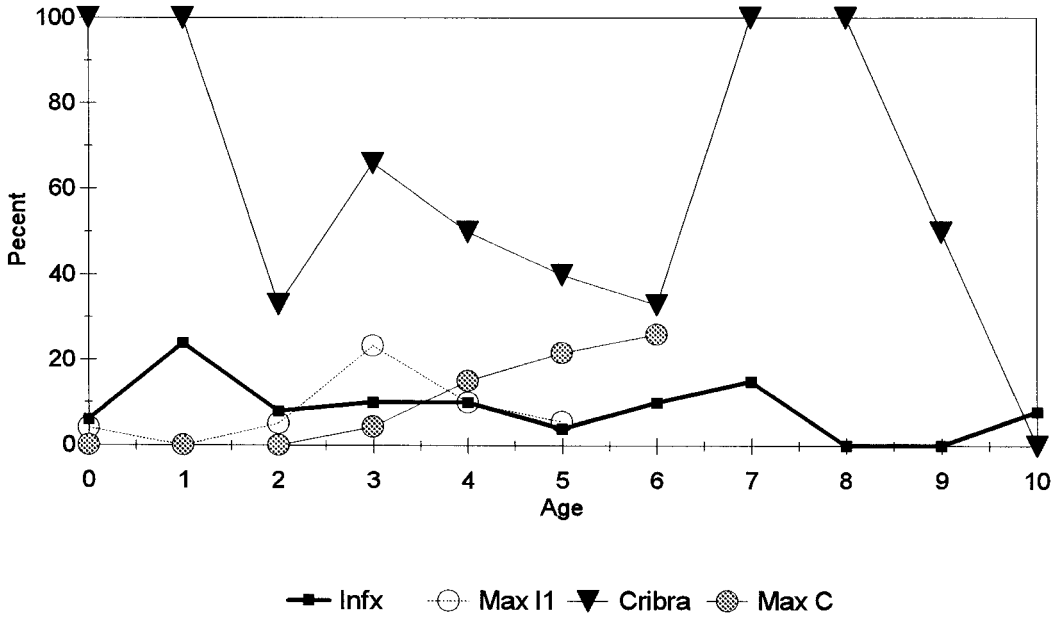

Fig. 8. LEH, infection, and cribra orbitalia.

may have lower social status than those with more males, and that those interred within latte set perimeters may have had higher rank. The within-latte and external burials clusters may also represent temporally separate groups. Graves (1991) suggests that the Tumon Beach communities are among the oldest and longest occupied on Guam; the space under the latte sets may have been exhausted, and other burial areas created in the later stages of the community's history.

People buried at Hyatt and other Tumon Beach sites might have been members of high ranking lineages, yet their children appear to have been more stressed than

TABLE 8. Mean numbers of hypoplasias in subadults with and without skeletal infection

Tooth	N	Mean LEH	Variance	*t*	D.F.	Significance[1]
Maxillary I1						
Infection	5	1.40	0.64	0.987	32	
No infection	29	0.72	1.23			
Maxillary I2						
Infection	4	1.00	0.50	1.042	29	
No infection	27	0.41	0.61			
Maxillary C						
Infection	3	2.67	0.22	1.976	25	.05
No infection	24	1.13	1.36			
Mandibular I1						
Infection	4	0.75	0.19	1.781	30	.05
No infection	28	0.21	0.17			
Mandibular I2						
Infection	4	1.50	0.75	1.857	29	.05
No infection	27	0.33	0.74			
Mandibular C						
Infection	4	1.75	2.19	1.686	19	.10
No infection	17	1.06	1.23			
All teeth						
Infection	10	7.90	83.09	2.208	95	.05
No infection	87	2.21	13.27			

[1] Significance is one-tailed.

those from the Apurguan site (see Douglas et al., 1997; Pietrusewsky et al., 1992). Compared to the Apurguan subadults, children in this sample have higher frequencies of LEH in deciduous teeth, more treponemal infection, and they had a higher probability of dying through age 14 years (Underwood, 1995). The substantial difference in LEH frequencies between adults and subadults is not seen in the Apurguan assemblage (Pietrusewsky et al., 1992; Douglas et al., 1997).

These apparent differences between the health of contemporaneous communities call for closer examination, as do possible intrasite differences in health patterns. Microclimatic change, the social organization of subsistence and settlement patterns, and the internal chronology of the 700-year-long Latte Period are current subjects of archaeological inquiry which will articulate well with the type of fine-grained paleoepidemiological analysis presented here.

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LITERATURE CITED

Ambrose SH, Butler B, Hanson DB, and Hunter-Anderson RL, and Kruger HW (1997) Stable isotopic analysis of human diet in the Marianas Archipelago, Western Pacific. Am. J. Phys. Anthropol. *104:*343–361.

Arriaza BT (1997) Spondylolysis in human prehistoric remains from Guam and its possible etiology. Am. J. Phys. Anthropol. *104:*393–397.

Barker DJP (1994) Mother, Babies, and Disease in Later Life. London: BMJ.

Berti PR and Mahaney MC (1992) Quantification of the confidence intervals for linear enamel hypoplasia chronologies. In AH Goodman and LL Capasso (eds.): Recent Contributions to the Study of Enamel Developmental Defects. Chieti, Italy: Journal of Paleopathology, Monographic Publication 2, pp. 19–30.

Bittner LH (1926) Some observations on the tertiary lesions of framboesia tropica, or yaws. Am. J. Trop. Med. *6:*123–130.

Blakeley ML and Armelagos GJ (1985) Deciduous enamel defects in Prehistoric Americans from Dickson Mounds: Prenatal and postnatal stress. Am. J. Phys. Anthropol. *65:*371–379.

Brewis AA (1995) Fertility and analogy in Pacific paleodemography. Asian Perspect. *34:*1–20.

Bruce-Chwatt LJ (1978) Yaws. In DB Jeliffe and JP Stanfield (eds.): Diseases of Children in the subtropics and tropics. London: Edward Arnold, pp. 800–812.

Condon K and Rose JC (1992) Intertooth and intratooth variability in the occurrence of developmental enamel defects. In AH Goodman and LL Capasso (eds.) Recent Contributions to the Study of Enamel Developmental Defects. Chieti, Italy: Journal of Paleopathology Monographic Publication 2, pp. 61–78.

Cook DC and Buikstra JE (1979) Health and differential survival in prehistoric populations: Prenatal dental defects. Am. J. Phys. Anthropol. *51:*649–664.

Douglas MT, Pietrusewsky M, and Ikehara-Quebral RM (1997) Skeletal biology of Apurguan: A precontact Chamorro Site on Guam. Am. J. Phys. Anthropol. *104:*291–313.

Driver M (1983) Fray Juan Pobre de Zamore and his account of the Mariana Islands. J. Pacific History *18:*198–216.

Driver M (1989) Fray Juan Pobre in the Marianas, 1602. University of Guam: Micronesian Area Research Center.

Duray SM (1996) Dental indicators of stress and reduced age at death in prehistoric Native Americans. Am. J. Phys. Anthropol. *99:*275–286.

Eason RJ, Somerfield SD, Tasman-Jones T, Henry A, and Jones G (1985) Resurgent yaws in the Solomon Islands. Aust. N. Z. J. Med. *15:*727–730.

Goodman AH (1996) Early life stresses and adult health: insights from dental enamel development. In CJK Henry and SJ Ulijaszek (eds.): Long-term Consequences of Early Environment: Growth Development and the Lifespan Developmental Perspective. Society for the Study of Human Biology Series No. 37. New York: Cambridge University Press pp. 163–183.

Goodman AH and Armelagos GJ (1985) Factors affecting the distribution of enamel hypoplasias within the permanent human dentition. Am. J. Phys. Anthropol. *68:*479–493.

Goodman AH and Armelagos GJ (1988) Childhood stress and decreased longevity in a prehistoric population. Am. Anthropol. *90:*936–944.

Goodman AH and Armelagos GJ (1989) Infant and childhood morbidity and mortality risks in archaeological populations. World Archaeol. *21:*227–242.

Goodman AH and Rose JC (1991) Dental enamel hypoplasias as indicators of nutritional status. In MA Kelley and CS Larsen (eds.): Advances in Dental Anthropology. New York: Wiley-Liss, pp. 279–293.

Goodman AH, Armelagos GJ, and Rose JC (1980) Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. Hum. Biol. *52:*515–528.

Goodman AH, Armelagos GJ, and Rose JC (1984) The chronological distribution of enamel hypoplasias from prehistoric Dickson Mounds populations. Am. J. Phys. Anthropol. *56:*259–266.

Goodman AH, Martinez C, and Chavez A (1991) Nutritional supplementation and the development of linear enamel hypoplasia in children from Solis, Mexico. Am. J. Clin. Nutr. *53:*773–781.

Goodman AH, Pelto GH, Allen LH, and Chavez A (1992) Socioeconomic and anthropometric correlates of lin-

ear enamel hypoplasia in children from Solis, Mexico. In AH Goodman and LL Capasso (eds.): Recent Contributions to the Study of Enamel Developmental Defects. Chieti, Italy: Journal of Paleopathology, Monographic Publication 2, pp. 373–380.

Grant MP, Knutsson I, and Stodder ALW (1992) Archaeological Monitoring and Data Recovery, AT&T Tumon Bay Cable Easement, Manhole, and Conduit System, Tumon, Tamuning Municipality, Territory of Guam. Prepared for AT&T Communications, Morristown, NJ. Paul H. Rosendahl, Ph.D. Inc., Hilo, HI. PHRI Report No. 91-1040.

Graves MW (1986) Organization and differentiation within late prehistoric ranked social units, Mariana Islands, Western Pacific. J. Field Archaeol. *13:*139–154.

Graves MW (1991) Architectural and mortuary diversity in late prehistoric settlements at Tumon Bay, Guam. Micronesica *24:*169–194.

Hackett CJ (1951) Bone Lesions of Yaws in Uganda. Springfield, IL: Charles C. Thomas.

Hackett CJ (1957) The transmission of yaws in nature. J. Trop. Med. Hyg. *60:*159–168.

Hanson DB (1988) Prehistoric mortuary practices and human biology. In BM Butler (ed.): Archaeological Investigations on the North Coast of Rota, Mariana Islands. Micronesian Archaeological Survey Report No. 23, Center for Archaeological Studies Occasional Paper No. 8. Carbondale: Southern Illinois University at Carbondale, pp. 375–435.

Hanson DB (1990) Paleopathological observations on human skeletal remains from Rota, Mariana Islands: Epidemiological implications. Micronesica Suppl. *2:* 349–362.

Hanson DB (1995) Mortuary and skeletal analysis of human remains from Achugao, Saipan. In BM Butler (ed.): Archaeological Investigations in the Achugao and Matansa Areas of Saipan, Mariana Islands. Saipan: Division of Historic Preservation Micronesian Archaeological Survey Report No. 30, pp. 311–343.

Henry CJK and Ulijaszek J (eds.) (1996) Long-term Consequences of Early Environment: Growth, Development and the Lifespan Developmental Perspective. Society for the Study of Human Biology Series No. 37. New York: Cambridge University Press.

Hill KR (1953) Non-specific factors in the epidemiology of yaws. First International Symposium on Yaws Control. Geneva: World Health Organization. World Health Organization Monograph No. 15, pp. 17–47.

Houghton P (1996) People of the Great Ocean: Aspects of Human Biology of the Early Pacific. New York: Cambridge University Press.

Hunter-Anderson RL (1995) Toward an explanation of diversity in Latte phase burials on Guam. Am. J. Phys. Anthropol. Suppl. *20:*114.

Hunter-Anderson RL and Butler BM (1995) An overview of Northern Marianas prehistory. Micronesian Archaeological Survey Report No. 31. Saipan: Historic Preservation Division, Commonwealth of the Northern Marianas.

Infante P (1974) Enamel hypoplasia in Apache Indian children. Ecol. Food Nutr. *2:*155–156.

Ikehara-Quebral R and Douglas MT (1997) Cultural alteration of human teeth in the Mariana Islands. Am. J. Phys. Anthropol. *104:*381–391.

Kreshover SJ (1960) Metabolic disturbances in tooth formation. Ann. N.Y. Acad. Sci. *85:*161–167.

Leigh RW (1929) Dental morphology and pathology of prehistoric Guam. Memoirs of the Bernice P. Bishop Museum *11:*1–18.

Malcom S (1955) Diet and Nutrition in the Trust Territory of the Pacific Islands. South Pacific Commission Technical Paper No. 83. Noumea: The South Pacific Commission.

Marples MJR and Bacon DF (1953) Observations on yaws and certain skin diseases in Manono, Western Samoa. Trans. R. Soc. Trop. Med. Hyg. *47:*141–147.

Massler M, Schour I, and Poncher HG (1941) Developmental pattern of the child as reflected in the calcification pattern of the teeth. Am. J. Dis. Child. *62:*33–67.

Martin DL, Goodman AH, Armelagos GJ, and Magennis AL (1990) Black Mesa Anasazi: Reconstructing Life from Patterns of Death and Disease. Carbondale: Southern Illinois University at Carbondale, Center for Archaeological Investigations, Occasional Paper No. 14.

Mills AR (1955) The incidence of yaws in the New Hebrides. Trans. R. Soc. Trop. Med. Hyg. *49:*58–61.

Pietrusewsky M, Douglas MT, and Ikehara-Quebral RM (1992) Prehistoric Chamorro Remains from the Apurguan Site, Tamuning District, Guam: An Osteological Investigation and Comparisons with other Micronesian Series. Honolulu: International Archaeological Institute, Inc.

Pietrusewsky M, Douglas MT, and Ikehara-Quebral RM (1997) An assessment of health and disease in the prehistoric inhabitants of the Mariana Islands. Am. J. Phys. Anthropol. *104:*315–342.

Pindborg JJ (1970) Pathology of the Dental Hard Tissues. Philadelphia: WB Saunders.

Rose JC, Armelagos GJ, Lallo JW (1978) Histological enamel indicators of childhood stress in prehistoric skeletal samples. Am. J. Phys. Anthropol. *49:*511–516.

Rothschild BM and Heathcote GM (1993) Characterization of the skeletal manifestations of the treponemal disease yaws as a population phenomenon. Clin. Infect. Dis. *17:*198–203.

Ryan EM (1995) Spatial distribution and mortuary procedure in the Marianas. Am. J. Phys. Anthropol. Suppl. *20:*187.

Sarnat B and Schour I (1941) Enamel hypoplasia (chronologic enamel aplasia) in relation to systemic disease: A chronologic, morphologic, and etiologic classification. J. Am. Dental Assoc. 28-1989-2000, *29:*67–75.

Simpson S, Hutchinson DL, and Larsen CS (1990) Coping with stress: Tooth-size, dental defects, and age at death. In CS Larsen (ed.): The Archaeology of Mission Santa Catalina de Guale: 2. Biocultural Interpretations of a Population in Transition. New York: Anthropological Papers of the American Museum of Natural History no. 68.

Stodder ALW (1987) The physical anthropology and mortuary behavior of the Dolores region Anasazi. In KL Petersen and J Orcutt (eds.): Dolores Archaeological Program Supporting Studies: Settlement and Environment. Denver: U.S. Bureau of Reclamation Engineering and Research Center, pp. 309–504.

Stodder ALW (1992) Human skeletal remains. In MP Grant, I Knutsson, and ALW Stodder: Archaeological Monitoring and Data Recovery, AT&T Tumon Bay Cable Easement, Manhole, and Conduit System, Tumon, Tamuning Municipality, Territory of Guam. Prepared for AT&T Communications, Morristown, NJ. Paul H. Rosendahl, Ph.D. Inc., Hilo, HI. PHRI Report No. 91-1040, pp. 38–45.

Stodder ALW (1993) Human Skeletal Remains from the Mangilao Golf Course Project, Mangilao Municipality, Territory of Guam. Paul H. Rosendahl, Ph.D., Inc. Hilo, HI. PHRI Project No. 89-744.

Stodder ALW (1994) Bioarchaeological investigations of protohistoric Pueblo health and demography. In CS Larsen and GR Milner (eds.): In the Wake of Contact: Biological Responses to Conquest. New York: Wiley-Liss, pp. 97–107.

Stodder ALW, Trembly DL, and Tucker CE (1991a) Human Remains from the Sandcastle Club Site. Paul H. Rosendahl, Ph.D., Inc. Hilo, HI. PHRI Project No. 89-882.

Stodder ALW, Trembly DL, and CE Tucker (1991b) Human Remains from the Camp Watkins Project. Paul H. Rosendahl, Ph.D., Inc. Hilo, HI. PHRI Project No. 89-757.

Stodder ALW, Trembly DL, and Tucker CE (1992) Paleoepidemiology and paleopathology of treponematosis in pre- and protohistoric villages in Western Micronesia. Am. J. Phys. Anthropol. Suppl. *14:*157.

Sutton PR (1978) Unusual forms of abrasion of teeth: Unintentional 'filing' in Micronesians and Polynesians. Archaeol. Phys. Anthropol. Oceania *13:*30–39.

Swardstedt T (1966) Odontological Aspects of a Medieval Population from the Province of Jamtland/Mid-Sweden. Stockholm: Tiden Barnangen, AB.

Thompson LM (1932) Archaeology of the Mariana Islands. Bernice P. Bishop Museum Bulletin 100. Honolulu: Bernice P. Bishop Museum.

Trembly DL (1995) On the antiquity of leprosy in western Micronesia. Int. J. Osteoarchaeol. *5:*377–384.

Trembly DL (1996) Treponematosis in pre-Spanish western Micronesia. Int. J. Osteoarchaeol. *6:*397–402.

White TD (1978) Early hominid enamel hypoplasia. Am. J. Phys. Anthropol. *49:*79–83.

Underwood JH (1973) Population history of Guam: Context of microevolution. Micronesica *9:*11–44.

Underwood JH (1995) Population history and dynamics: Prolegomenon to Marianas skeletal biology. Am. J. Phys. Anthropol. Suppl. *20:*214.